Aus der Neurologischen Universitätsklinik Basel
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60 Cases of Incidental Findings of Subclinical Ulnar Nerve Lesions

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Introduction

The ulnar nerve is one of the most frequently injured of the peripheral nerves in the general population as well as in war injuries. Within the group of traumatic nerve lesions, it is the most common lesion of all.[1]

If the injury is caused by a sudden trauma, the etiology and location of the lesion are usually known. When however the injury develops slowly with a minimum of clinical symptoms, the diagnosis is more difficult and often overlooked. The interval between the injury and the clinical examination varies widely, the interval sometimes being sufficient for contractures to develop or for the patients to learn trick movements, for example various manipulations to compensate the loss of strength of certain muscles.[2]

The ulnar nerve does not give off any branches above the elbow. In the forearm it supplies the flexor carpi ulnaris, the medial part of the flexor digitorum profundus, the muscles of the hypothenar eminence, the interossei, the lumbricals to the little and ring fingers and the adductor pollicis. Its sensory distribution usually comprises the little finger and the ulnar half of the ring finger, and corresponding parts of the hand to the level of the wrist.[3]

One of the first reports in the literature describing the clinical picture of ulnar paralysis was given by GRANGER in 1818.[4] CALDER in 1832 described some of the clinical features of ulnar nerve paralysis due to an injury at the level of the sulcus.[5] DUCHENNE in 1867 was one of the first to analyse the causes of claw hand.[6]

Symptoms vary with localisation of the injury. At the elbow, pressure lesions often give rise to aching pain, mostly on the ulnar side of the joint with some radiation proximal and distal. There are also paraesthesiae in the little finger. Gradually weakness develops in the hand particularly on flexion of the little and ring finger. Very often the first symptom of an ulnar lesion is the atrophy of the small muscles of the hand. Complete ulnar paralysis results in a characteristic claw hand deformity owing to wasting of the small hand muscles and hyperextension of the fingers at the metacarpophalangeal joints and flexion at the interphalangeal joints.

Froment's sign is one of the more constant symptoms of ulnar paralysis. The patient is unable to hold a piece of paper between the stretched thumb and the palmar aspect of the forefinger, and has to flex the thumb for lack of strength of the adductor pollicis. It appears in advance of many more obvious signs of clinical ulnar nerve paralysis, and often remains for a long period of time during the recovery of function. Among 200 cases of clinical ulnar nerve paralysis, BABINSKI found it only twice negative. It is by simultaneous ulnar and median nerve paralysis negative.[7] MUMENTHALER[8] reported on a large number of cases of ulnar paralysis. He has made special mention of two groups, one of which is caused by a single direct trauma and results in an acute ulnar paralysis and a second group which occurs with an incidence more than twice that of the first in which the paralysis developes only after a prolonged series of trauma. This second group he has called non-direct traumatic ulnar paralysis. This expression non-direct trauma, represents lesions in which there is no severing and no direct immediate damage to the nerve. This is one reason why many cases are classified as of uncertain etiology.

Among the injuries caused by direct trauma are included fractures, luxations particularly in the elbow and wrist, and direct severing of the ulnar nerve.

Among the non-direct trauma, tardy ulnar paralysis can be caused by fractures of the medial or the lateral condyle, dislocation of the elbow and changes due to arthritis in the elbow joint. The fracture may be complicated by a cubitus valgus. A chronic trauma consisting of pressure or stretching of the nerve will lead to ulnar damage. At the wrist the nerve may be injured by lacerations.[9]

The entrapment syndromes of the ulnar nerve can best be understood by studying its topography. The ulnar nerve is most commonly affected where it passes behind the medial epicondyle of the humerus superficially. Here it is very frequently damaged by intermittent pressure particularly if the sulcus is shallow and the nerve is therefore easily displaced from it. It can also be affected immediately below this where it passes between the heads of origin of the flexor carpi ulnaris muscle. At the wrist the ulnar nerve may be compressed in passing through the loge of Guyon which is on the radial side of pisiform underneath part of the transverse carpal ligament, and in the hand when it passes beneath the origins of the hypothenar or adductor pollicis muscles. Nerve lesions at these points are usually precipitated by vigorous muscular activity.[10]

In certain occupations involving the use of vibrating tools, the repeated pressure on the proximal part of the hypothenar eminence can lead to damage. This problem of occupational trauma to the ulnar nerve is widespread. Craftsmen such as jewelers, engravers and machinists who hold the tools tightly and exert hard pressure in the palm suffer injury to the deep palmar branch of the ulnar nerve.[11] Other occupations that lead to ulnar damage are foundry ladlers, telephone operators, and motorists who rest their arms on the car window ledge. Carpentry, which requires frequent flexion and extension of the elbow, subjects the ulnar nerve to repeated minor injury.

Most of these patients show one or more of the symptoms ascribed to ulnar nerve lesions. There is however a group of patients whose symptoms are more diffuse, and others who do not present with symptoms.

The measurement of conduction velocity is helpful in the assessment and accurate location of minor ulnar nerve lesions. By stimulating the nerve at different points along its course, the conduction rate for different segments, can be evaluated separately (12, 13, 14, 15, 16, 17).

Another valuable help is the recording of the sensory action potential of the ulnar nerve by stimulating the digital nerves and recording the action potential from the nerve trunk at the wrist (18, 19).

In peripheral nerve lesions such as carpel tunnel syndromes, by pressure lesions of the ulnar nerve at the elbow, and in neuritis and polyneuritis, the sensory nerve action potential disappeared early or appeared with a prolonged latent period. This can also be shown even when there are no objective clinical signs of sensory disturbances (20).

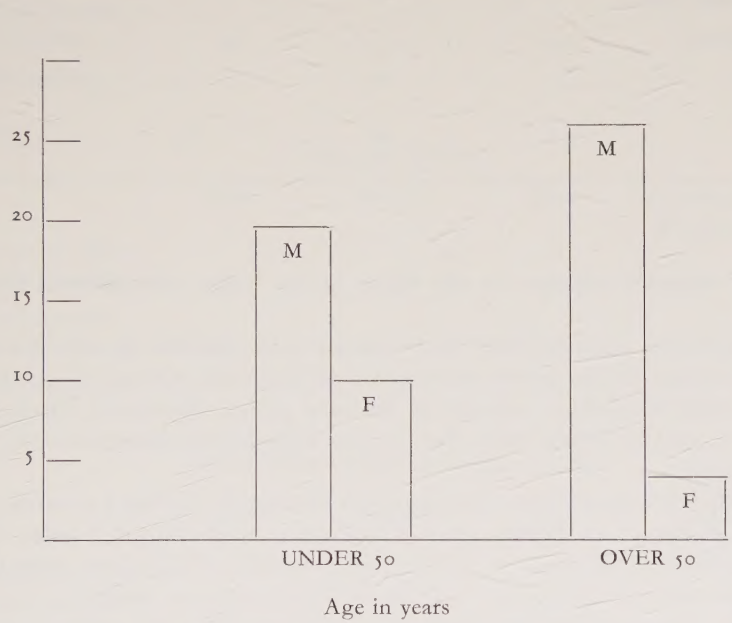
With the methods of motor and sensory nerve conduction measurements, many cases of ulnar nerve lesions are detected by chance. These patients have either no symptoms or complain only, when they are questioned about paraesthesia on the little finger or about sensitivity of their elbows.

The aim of this study is to analyse the cases of these subclinical ulnar nerve lesions.

The case histories of 298 patients with impairment of ulnar nerve conduction who were seen at the Basel Neurological Institute, were reviewed. 60 cases were detected incidentally by electromyography, when looking for carpel tunnel syndromes or when comparing the apparently healthy side with the ulnar nerve lesion on the other side, and in cases of diffuse brachial pains.

Figure 1 shows the distribution of the incidental ulnar nerve lesions by sex and age. Among the 30 cases under 50 years, 20 were male and 10 were female giving a male to female ratio of 2:1. In the group over 50 years of age, 26 were male and 4 were female, giving a male to female ratio of 6.5:1.

Fig. 1. 60 Cases of Subclinical Ulnar Nerve Lesions Showing Distribution by Sex and Age



Once the diagnosis of a subclinical ulnar nerve lesion was established by electromyography, we searched for possible causes or coincidental findings. The results are given in Table 1. It further shows the distribution by sex of the findings. There were 10 cases of subluxation of the ulnar nerve in the ulnar groove, of which 9 were male and one was female. Of 5 cases due to chronic pressure, 2 were male and 3 were female. 3 cases were due to chronic occupational trauma. Coincidental Carpel Tunnel Syndrome was present in 11 cases. Of these, 7 were female and 4 were male. There were 3 cases of Dupuytren's contracture and one coincidental traumatic plexus brachialis lesion. Also, 1 case of diabetic neuritis was found. In almost half the cases, the nature of their

TABLE 1

Etiology and Concomitant Diseases in 60 Patients with Subclinical Ulnar Nerve Lesions

Diagnosis	Male	Female	Total
Subluxation of the Ulnar Nerve	9	1	10
Chronic pressure	2	3	5
Trauma chronic	3		3
Carpel Tunnel Syndrome	4	7	11
Dupuytren's Contracture	3		3
Plexus Brachialis Lesion	1		1
Diabetic Neuritis	1		1
Unknown	23	3	26
Total	46	14	60

ulnar lesion remained obscure. In the whole group, males outnumbered females by 46 to 14.

Table 2 shows the types of EMG abnormalities in the different groups of subclinical ulnar nerve lesions. In the group with known or supposed etiology 65.9% showed a slowing in motor conduction velocity. In the same group, the sensory action potential was absent in 29.5%. There were also 2 cases with partial denervation of the first interosseus muscle.

In the group of incidental lesions of unknown etiology 61.3% had a slowing in motor conduction velocity of which there were 16 findings in males and 3 in females. In 35.5% there was absence of the sensory nerve action potential, all of which were in males. 1 patient presented a partial denervation of the first interosseus muscle.

In Table 3, I have shown the localisation of the slowing of conduction velocity. In the group of known or supposed etiology 89.7% of the lesions were in the elbow. There were two males with an upper arm lesion, and another with a lesion in the forearm. Under lesions of unknown etiology, in all but 1 patient the lesion could be located in the elbow region. The combined totals show that 91.7% of all incidental lesions were supposed to be located in the elbow. 3 males had lesions in the upper arm and one other male in the forearm.

Table 4 shows the values of the motor conduction velocities in the different segments of the ulnar nerve of those patients having findings, compared with a control group of 100 healthy subjects. In the cases with nerve lesions in the upper arm the mean average velocity was 40.7 m/sec against a normal average of 63 m/sec. The values in the elbow were also reduced, being 51.3 m/sec against a normal average of 60 m/sec.

In the cases suggestive of ulnar nerve damage in the elbow, we found an average velocity of 41 m/sec against a normal average of 60 m/sec. In the one case with a forearm lesion, the velocity in the forearm was 32.5 m/sec against a value of 55 m/sec for the control group.

TABLE 2

EMG Findings in 60 Cases of Subclinical Ulnar Nerve Lesions

	Known Etiology			
	Male	Female	Both	% of Findings in Cases of Known Etiology
Disturbance in motor Conduction Velocity	19	10	29	65.9
Absence of Sensory Action Potential	10	3	13	29.5
Partial Denervation	2	–	2	4.6

	Unknown Etiology			
	Male	Female	Both	% of Findings in Cases of Unknown Etiology
Disturbance in Motor Conduction Velocity	16	3	19	61.3
Absence of Sensory Action Potential	11	–	11	35.5
Partial Denervation	1	–	–	3.2

	Totals of Known and Unknown Etiology			
	Male	Female	Both	% of Findings in Cases of Known and Unknown Etiology
Disturbance in Motor Conduction Velocity	35	13	48	64.0
Absence of Sensory Action Potential	21	3	24	32.0
Partial Denervation	3	–	3	4.0
Totals*	59	–	–	78.6
		16	–*	21.4
			75	

* Number of Findings exceed total number of patients. Many patients have more than one incidental finding.

TABLE 3

Localisation of Subclinical Ulnar Nerve Lesions with Conduction Velocity Disturbances

	Known Etiology			
	Male	Female	Both	% of Lesions of Known Etiology
Upper arm	2	–	2	6.9
Elbow	16	10	26	89.7
Forearm	1	–	1	3.4
	Unknown Etiology			
	Male	Female	Both	% of Lesions of Unknown Etiology
Upper arm	1	–	1	5.3
Elbow	15	3	18	94.7
Forearm	–	–	–	
	Totals of Known and Unknown Etiology			
	Male	Female	Both	% of Lesions of Known and Unknown Etiology
Upper arm	3	–	3	6.2
Elbow	31	13	44	91.7
Forearm	1	–	1	2.1
Totals	35	–		72.9
		13		27.1
			48	

TABLE 4

Motor Nerve Conduction Velocities – Incidental EMG Findings

	No. of Cases	Latency distal	Conduction Velocities m/sec		
			Forearm	Elbow	Upper arm
Normal Control Group	100	2.5 (2.7–3.4)	55 (49–64)	60 (53–73)	63 (54–76)
Lesions in					
Upper arm	3	3.4 (2.8–4.1)	68.5 (65–72)	51.3 (46–56)	40.7 (33–45)
Elbow (Sulcus)	44	2.9 (2.0–4.6)	59.1 (42–87)	41 (17.5–51)	64.3 (44–91)
Forearm	1	1.6	32.5	58.5	52.5

Conclusions

60 cases of subclinical ulnar nerve lesions were found, among a total group of 298 patients with ulnar nerve lesions. This represented 20.1% of all patients with a diagnosis of ulnar nerve lesion on the basis of the EMG findings.

The elbow was found to be the site of 91.7% of all of the incidental lesions found. In the largest group of patients, the cause of the lesion at the elbow could not be detected. Males accounted for 76.7% of the patients with incidental findings, females 23.3%. The male to female ratio in the group over 50 years of age was 6.5 to 1, of the group under 50 years the ratio is only 2 to 1.

The diagnosis of a subclinical ulnar nerve lesion was based on the following EMG findings: a slowing of the motor nerve conduction velocity in one segment of the nerve, the absence of the sensory nerve action potential or the recording of fibrillation potentials in the first interosseus muscle in the absence of finding suggesting a polyneuritis, plexus or anterior horn cell lesion.

The results of our study support the clinical impression that the ulnar nerve, particularly in the region of the elbow, is more vulnerable than other nerves. In the ulnar groove the nerve is exposed to compression and to repeated minor traumatisations, especially in cases of anatomical variations of the sulcus and of subluxation of the nerve. For these reasons it is not surprising to find a slowing of conduction velocity or an absence of the sensory nerve action potential on the apparently healthy side when we examine a patient with a severe ulnar lesion on the other side. In cases of a subclinical ulnar nerve lesion, a minor accident, excessive elbow movements, prolonged bed rest or supporting the head while sitting at a table, may provoke the clinical manifestations of an ulnar palsy. In order to evaluate the significance of a trauma as a cause for an ulnar nerve palsy, it is necessary to examine also the other ulnar nerve with the methods of measurement of the motor and sensory nerve condition velocity and needle EMG.

After trauma to the elbow, ulnar nerve damage may thus be detected well ahead of the clinical manifestations of a tardy ulnar palsy. In those cases repeated EMG examination will show, whether the nerve lesion is progressive and necessitates surgical intervention before the appearance of the claw hand.

Summary

(1) The case histories of 298 patients with impairment of ulnar nerve conduction were reviewed.

(2) Among these, 60 cases of subclinical ulnar nerve lesions were found coincidentally, with the methods of measurement of the motor and sensory nerve conduction velocity and the needle EMG.

(3) The results of our study support the clinical impression that the ulnar nerve, particularly in the region of the elbow, is more vulnerable than other nerves.

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Curriculum vitae

Am 5. November 1932 wurde ich in Boston (Massachusetts) U.S.A. geboren. Ich habe die Primarschule und die Bronx High School of Science in der Stadt New York besucht. An der New York University erwarb ich im Juni 1953 das Bachelor of Arts Diplom. Ich bestand das zweite Propaedeuticum in Basel, 1961. Meine klinischen Studien absolvierte ich in Basel. Im Dezember 1965 schloß ich meine Studien mit dem Staatsexamen in Basel ab.